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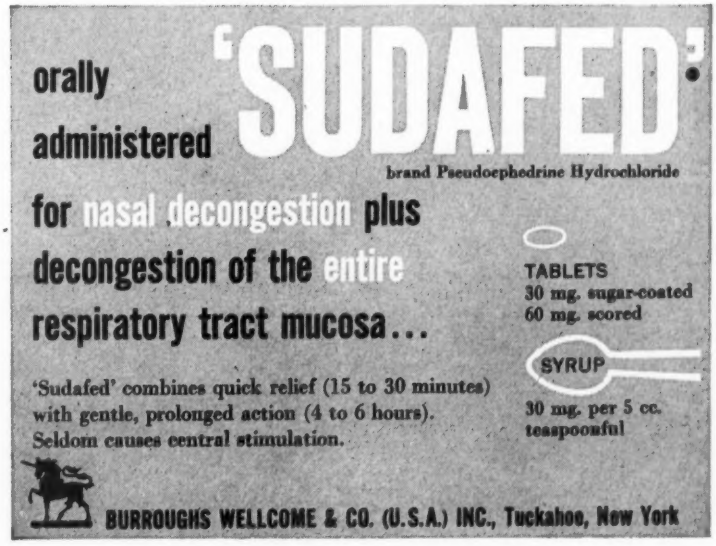
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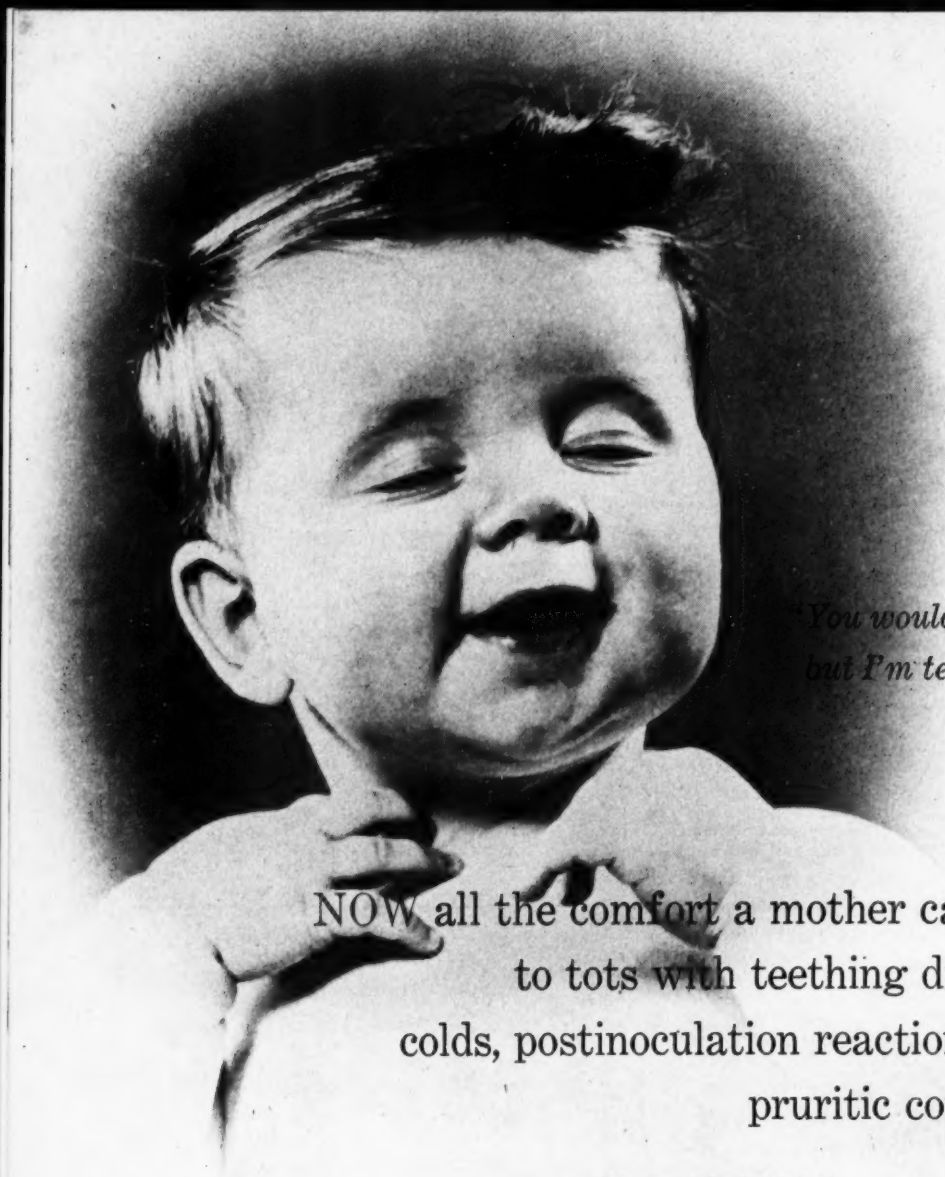

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SOUTHWESTERN MEDICINE

VOL. XL FEBRUARY, 1959 No. 2

Hypotensive Drugs

By Lester C. Feener, M.D., F.A.C.P., El Paso

In recent years new drugs which really reduce blood pressure have become universally used. Prior to this time our treatment of hypertension left a great deal to be desired. The use of these drugs is not particularly intricate when individualized. However, there are numerous general considerations which must be investigated prior to the treatment of any hypertensive.

Let us ask ourselves the question as to who should be treated for hypertension. All patients whose diastolic blood pressure remains above ninety millimeters of mercury should at least be investigated and considered as candidates for treatment until the necessity for treatment has not been proven. Here it well may be said that the very mild cases do not need intensive therapy.

Perhaps weight reduction, psychotherapy, such drugs as sedatives, especially phenobarbital, and tranquilizers may, at least at the beginning, suffice insofar as therapeutic procedure is concerned. The old conception that blood pressure was "essential", I believe, has been disproven. It is possible that hypertension might be considered to be "essential" in advanced renal disease where the intraglomerular pressure is exceedingly high. It might be better if the term "essential hypertension" were discarded.

Individualization

While cases of hypertension have factors in common, its treatment must be individualized. This individualization requires separate evaluation with each patient. This evaluation and adequate history is important. The physician should know his patient. He should know, for example, the type of work the patient does; his home life; and factors contributing to any emotional instability he might have. He should know how well the patient is able to adjust to the factors themselves.

The physical examination must be complete and have adequate laboratory procedures in order to evaluate blood vessel structure. The blood vessel structure of the fundi, kidneys, coronary vessels, and cerebral vessels are of prime importance. Stressing again individuality in the treatment for purposes of therapy, it becomes evident for example, that one cannot treat an arteriosclerotic patient, say 70 years old or more, in the same manner one treats the younger patient. Indeed in these elderly sclerotic patients, one may do more harm treating the patient, especially with certain drugs, than good by blood pressure reduction. There are certain elderly patients who are perhaps better left alone with a mild or moderate degree of hypertension.

Special consideration must be given to the patient with nitrogen retention due to renal disease because intensive therapy may possibly increase the nitrogen retention and even cause a renal shutdown. Remember, while anti-hypertensive therapy may actually reverse to some degree the "strain pattern" one sees on the electrocardiographic tracing, the therapy may actually aggravate the anginoid

*Dr. Feener this month is substituting for Dr. Jack A. Bernard, regular conductor of this section.

CURRENT THERAPY

phenomena one sees in these patients.

For example, it is felt by many that ganglionic blocking agents are contra-indicated with the anginoid phenomena present, and that one should treat these patients with, let us say, rauwolfia, veratrum, or chlorothiaxide, or some combination of these drugs.

Blood Pressure Determination

Let us consider briefly the blood pressure determination itself. It must be realized that the patient who is definitely hypertensive in your office may be a normal tensive at home. As one becomes better acquainted with the patient, it becomes easier to determine the normal for that particular patient. Whether some member of the family should be taught to take blood pressure determinations in all cases is debatable.

There can be no doubt that home blood pressure determinations are a necessity in cases in which ganglionic blocking agents are required. Since the advent of cholothiazide, the use of these agents has decreased; hence the necessity of home blood pressure determinations has been reduced proportionately.

Usually if the patient is not hurried, and the patient and physician approach the problem at hand in a leisurely and friendly manner, blood pressure determinations can be made adequately in the office. It must be admitted, however, that there are exceptions to this, and that the emotionally unstable hypertensive may require home blood pressure determinations as a therapeutic guide.

Lastly, how effective are anti-hypertensive drugs. It was said before the advent of chlorothiazide, they were about fifty per cent effective. Since the introduction of chlorothiazide, their effectiveness has been increased. However, there are so many ancillary factors in the treatment of hypertension, that the percentage evaluation can never attain exactitude.

Texas Postgraduate Meeting Scheduled in Fort Stockton

Obstetrics and Gynecology will be the subject of a one-day meeting of the El Paso Division of the University of Texas Postgraduate School of Medicine, to be held from 9 a.m. to 4:30 p.m., Thursday, February 12, 1959, in the Community House in Fort Stockton, Texas.

Physicians from West Texas, New Mexico and northern Mexico are invited to attend. Seven hours of credit will be given. Dr. Ralph H. Homan of El Paso is Director of the El Paso Division.

The program is as follows:

8:30 - 9:00 a.m.	Registration
9:00 - 9:50 a.m.	Functional Uterine Bleeding. Dr. S. Leight Avner, El Paso
9:50 - 10:00 a.m.	Intermission
10:00 - 10:50 a.m.	Practical Points in Gynecological Pathology; Dr. Charles L. Green, El Paso.
10:50 - 11:40 a.m.	Endocrinology in Obstetrics and Gynecology; Dr. Harry W. Demarest, El Paso
1:00 - 1:50 p.m.	Repeat Cesarean Section. Dr. James R. Morgan, El Paso
1:50 - 2:40 p.m.	Minimal Diagnostic Surgery in Infertility. Dr. Gray E. Carpenter, El Paso.
2:40 - 2:50 p.m.	Intermission
2:50 - 3:40 p.m.	The Management of Carcinoma of the Female Genital Tract; Dr. Felix M. Rutledge, Houston, Texas.
3:40 - 4:30 p.m.	Reconstructive Pelvic Surgery. Dr. Gilbert Landis, El Paso.

(Each lecture is to be forty minutes long, followed by a ten-minute question and answer period.)

W. Compere Basom, M.D., *El Paso, Orthopaedic Editor*

ORTHOPAEDIC SURGERY NOTES

How to "Pin" a Hip and/or Internally "Fix" an Intertrochanteric, Subtrochanteric or Comminuted Fracture of the Upper Third of the Femoral Shaft

Permission was granted by the Journal of International College of Surgeons to re-print the following illustrations from the Journal of International College of Surgeons, Vol. XXV, No. 3, Mar. 1956.

Figure 2-A. Reveals the skin incision to expose the upper femur. This incision begins at the anterior superior spine. It curves posteriorly and inferiorly over the posterior aspect of the greater trochanter and then extends distally.

Figure 2-B. Reveals the deep fascia incised. There is a translucent membrane which connects this fascia to the trochanter. If this is divided at the area where the retractor is shown, the incision can be extended superiorly and somewhat anteriorly so that the operator can easily identify the interval between the gluteus medius which is shown in the upper posterior part of the incision and the tensor fasciae latae which is in

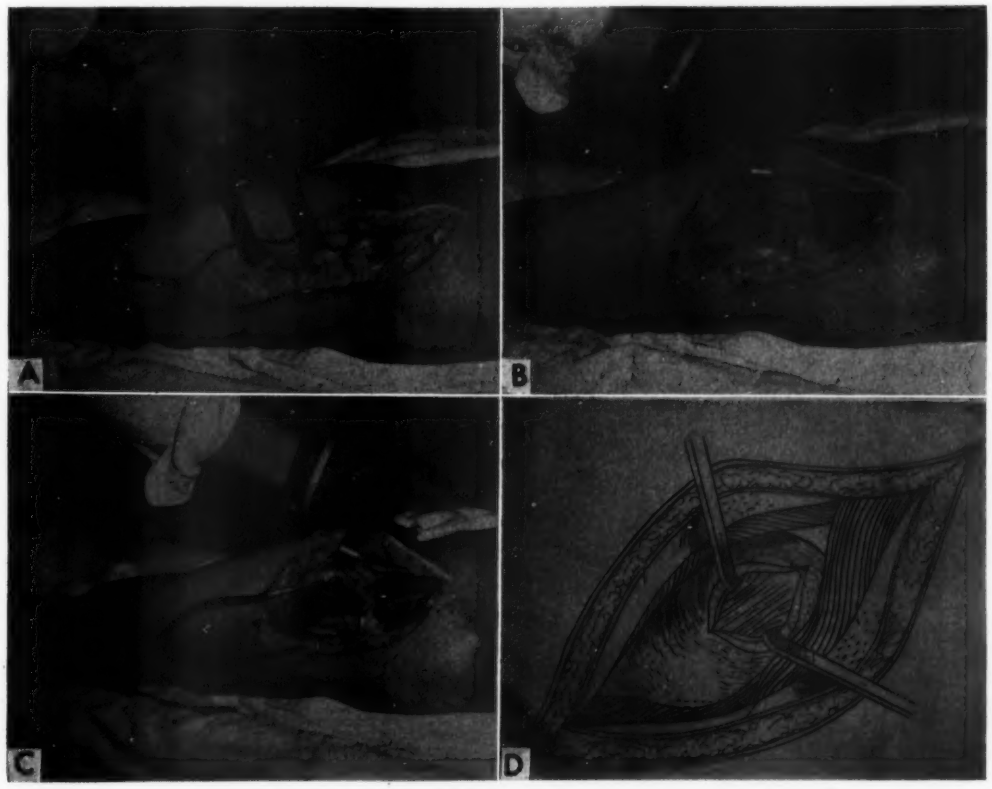

Figure 2—A, B, C, D

the anterior part of the incision; and the femoral neck covered by the capsule is easily exposed.

Figure 2-C. Reveals the incision in the vastus lateralis to expose the trochanteric region. An opening is made here for the guide pin and insertion of the nail. This incision can be extended distally as far as desired so that intertrochanteric fractures can be fixed in this manner after the

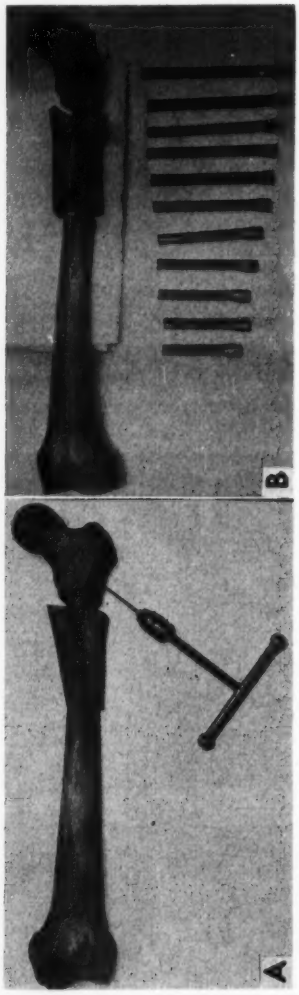

Figure 3—A, B

nail has been inserted. By the use of variable lengths of plates the fractures of the subtrochanteric and upper third shaft of the femur can be fixed.

Figure 2-D. Shows the capsule incised to expose the femoral neck and with this incision a

Figure 4—A, B

femoral neck fracture can be easily exposed. This exposure gives the operator the advantage of removing bits of infolded periosteum and flakes of cortical bone located transversely across the fracture site. The fracture can be accurately reduced. The guide pin of course can be inserted under direct vision, and the femoral head can be derotated into proper position. After fixation the fracture site can be tested. It is amazing how many of these still have instability following excellent fixation. With these cases additional fixation such as a piece of cortical bone through a drill hole across the fracture site can be utilized.

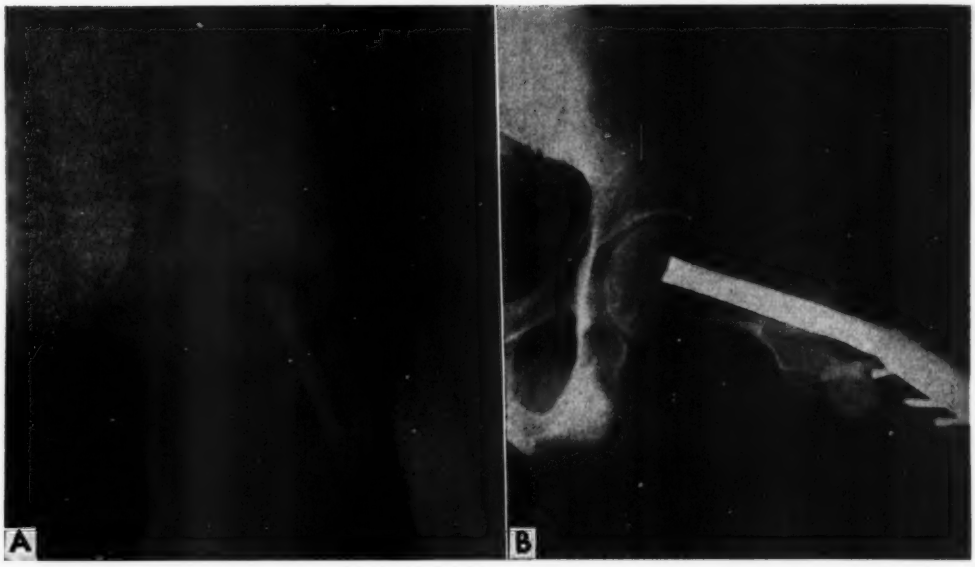

Figure 5—A, B

Figure 3-A. Reveals the hole made in the bone and a guide pin being inserted under direct vision by the use of a hand chuck.

Figure 3-B. Reveals the various lengths of stainless steel cannulated Smith-Petersen type nails. These nails have a serrated head to fit in a serrated well to the trochanteric plate.

Figure 4-A. Shows the fracture reduced, the plate applied. A bolt used for connecting the plate to the nail lies just distal to the T-wrench used for tightening the bolt. The bolt has a guide pin on its tip which easily guides it into proper position.

Figure 4-B. Shows x-rays of an anatomical study. It is better to reduce the fracture, then apply the plate, than it is to apply the plate and then have the plate in the way of the reduction. Also the plate can easily be put into the distal part of the wound first and then anchor it to the nail. In that way the soft tissue incision and exposure is much less extensive.

Figure 5—A, B, C

Figure 6

Figure 5-A, B, C. Shows extremely comminuted fracture of upper third of left femur. Lateral before surgery; AP and lateral after surgery.

Figure 5-A & B. Shows flexed lateral view, before and after surgery; after use of nail plate apparatus. (Courtesy of Dr. Mario Palafox)

Figure 6. Anteroposterior film of fracture shown in Figure 5-A, taken three months after reduction and fixation.

This study was reported in Yearbook of Orthopaedics and Traumatic Surgery 1956-57 Series, The Yearbook Publishers, Inc., Dr. Edward L. Compere the Editor, stated that many failures in the treatment of comminuted fractures of the upper third of the femoral shaft and intertrochanteric area have resulted from the use of a plate too short to be securely attached to the distal intact portion of the femoral shaft. "The extra-long plate with eight or more screw holes or slots does give excellent fixation for maintaining reduction and alignment."

Communicable Disease Workshop Scheduled in Albuquerque

A workshop on communicable disease control will be held in Albuquerque, Apr. 7-9, according to announcement by Dr. John Mason of the New Mexico Health Department. The workshop will deal primarily with operational problems of communicable disease control in the field, and all interested public health workers are invited to attend.

The U. S. Public Health Service Communicable Disease Center in conjunction with the state division will present the workshop with the following topics to be discussed: enteric infections, food poisoning, diphtheria, encephalitis, tuberculosis, venereal diseases, infectious hepatitis, staphylococcal infections in hospitals, polio, streptococcal infections and rheumatic fever, rabies control and the handling of animal bites, and immunization procedures.

The workshop has been timed to follow the annual meeting of the New Mexico Public Health Association which will be held in Albuquerque, Apr. 6 & 7.

Coming Meetings

University of Texas Postgraduate School of Medicine, Temple Division, Seventh Postgraduate Conference in Medicine and Surgery, sponsored by Scott, Sherwood and Brindley Foundation, Temple, Tex., Mar. 2-4, 1959.

University of Colorado Medical Center, Denver, Colo., Postgraduate Course, Medical Technology, Mar. 16-21, 1959.

Biennial Western Conference on Anesthesiology. Approved for 24 hours Category II Credit, A.A.G.P. Westward Ho Hotel, Phoenix, Apr. 1-4, 1959. For information write to Boyden L. Crouch, M.D., 301 W. McDowell Road, Phoenix, Ariz.

The Arizona Medical Association, 68th Annual Meeting, San Marcos Hotel, Chandler, Ariz., April 29-May 2. Friday, May 1, has been designated as Medical Education Day.

New Mexico Medical Society, annual meeting, Las Cruces, N. Mex., May 5-7, 1959.

Southwest Obstetrical and Gynecological Society, Annual Meeting, El Mirador Hotel, Palm Springs, Calif., Nov. 2 and 3, 1959.

Southwestern Medical Association, Annual Meeting, Roswell, N. M., Nov. 5-7, 1959.

The Management of Respiratory Allergy*

By George S. Richardson, M.D., Roswell, N.M.

This subject is undoubtedly one of considerable controversy and, judging from my experience with some of the end-results of respiratory allergies, of considerable import. It is a difficult subject to cover adequately. A patient is sometimes equally difficult to manage or benefit.

James[1] states that Bostock of Guy's Hospital wrote the first treatise on "Hay Fever" under the title of "Summer Catarrh" in 1828. He also quotes the interesting work of Leavitt on 5,000 inmates of mental institutions and epileptics with no trace of asthma being found in that number. James feels that intellectuals are affected more frequently than working people, particularly those who do hard physical work. This factor is undoubtedly corroborated in our own daily practice to some degree.

James further quotes Vaughn's figures that 10 per cent of Americans are frankly allergic and that 50 per cent of us give some history of transient episodes of allergy. An interesting old Latin proverb. "Quod cibus est aliis, aliis, est atre venenum" ("What is food to some is black poison to others.") is borrowed for this paper. Hilsinger[2] quotes a table by Vaughn and Black showing that between three and four per cent of an entire community of 508 persons suffered from asthma in their study.

For The Record

For the purpose of the record we shall consider nasal congestion, pharyngitis, laryngitis, tracheo-bronchitis, certain atypical pneumonias and asthma as being definite forms of respiratory allergy. I have never been able to distinguish an intrinsic asthma from the other types. Despite the feeling in certain quarters that emphysema is a definite clinical entity, one wonders at times if this and certain other pathological entities are not the sequelae of the asthma.

It is my feeling that the management of respiratory allergy depends upon two things. They are early recognition and the institution of measures of desensitization to or removal of the offending allergen.

In all instances the cure is not effected this simply and the removal of the offending allergen is often difficult. The pediatrician and the oto-laryngologist, of the specialties, are first to see the bulk of allergies. The general practitioner sees all types first.

Everyone Allergic

One wonders where all of this allergy was twenty years ago. Allergy, or the tendency toward allergy follows Mendelian laws. The growth of our population, as it currently progresses (something like the current stock market), will result in a population at one phase in which all members of society are allergic.

At present we can say that the newborn babe who wheezes after nursing has been sensitized as a fetus through the mother and is probably our first asthmatic. The great bulk of allergies may be in relation to feeding problems as new foods are added to the diet. Eighty per cent of allergies in children are probably the same as their parents, and all of these may be the same because of the eating habits in the family.

In the first nine months of life one sees eczemas, colic and diarrheas. These may be due to dust and foods, but the habit of using a food addition meal after meal is a prime cause for trouble. Between the ages of twelve to eighteen months we have more asthmas. Patients do not have to be a

*Delivered at Southern New Mexico Clinical Meeting, Artesia, November 23, 1958.

certain age before they can be sensitive to specific inhalants. The marasmic baby may be sensitive to dust. The age of six to ten years include the patients breaking down to foods while the twelve to fifteen year olds include the pollen sensitive. These last are the ones who "grew out of their trouble". Adults break down in their late thirties or early forties and develop clinical allergies for the first time—for as yet some unknown reason.

It is felt in certain quarters that the infants with eczema become your hay fever and asthmatic patients. Rinkel[3] has stated that his oldest proven allergy patient is eighty-four years of age. This is a reasonable concept. Imagine eating the same old breakfast for eighty-four years without trouble! For those who include the "nerves" in the etiology, the morning newspaper is filled with much cause for "soul searching".

We are primarily concerned here with pollen inhalants, the non-pollen inhalants (dust, animal danders and mold spores) and food allergens. They may act singly or concomitantly. They are affected by thermal sensitizations. Some of the non-pollen inhalants are removed as simply as taking pet skunks, for example, out of the house.

The pathology of all allergy consists of (1) smooth muscle spasm, (2) increased capillary permeability and edema and (3) eosinophilia. The absence of eosinophilia, whether in peripheral blood or nesal smear, is certainly no real diagnostic criterion for saying that no allergy exists. And, knowing this basic pathology, one may understand the physiology or pathologic physiology of the symptoms which confront the examining physician who sees a severe allergic individual. Your tight watch bands this morning which progressively get tighter as the day progresses may indicate that you have eaten well—if not wisely.

Primary Request

One does not ask that you go "whole hog" for this subject of allergy but primarily asks that you consider allergy in your differential diagnosis and rule out everything else from ulcer crater to rectal polyp. As an example, it would be devious to test the urine of all asthmatics with the qualitative test for 5 hydroxy-indoleacetic acid (5 HIAA) as described by Udenfriend[4] for the carcinoid syndrome. The magnitude of the task confronting medicine is hard to comprehend but demands more than skepticism or actual disbelief by the best of our profession.

Remember the specificity of the shock organ—the organ or tissue in which reaction occurs. It can be highly localized—left upper lid to dorsum of the right foot. Aside from the respiratory phase, one old lady from Artesia has been recently seen who had a trigeminal neuralgia handled by alcohol injection with good and lasting relief. Her recurrences have been handled with fractional house dust treatment and this appeals to me more than the alcohol treatment.

One must continually take a good history. This is the basis for good medicine and a "good" physician. One must not, in considering the allergy, believe the individual, who gets up in the morning and spits, sneezes and coughs, particularly sensitive to her kitchen nor his shaving cream. This, as Rinkel states, is simply the sum total of yesterday's allergic insults.

Dust and Mold Spore

Patients in the dust and minor mold spore category are fixed there by certain characteristic symptoms. The dust-sensitive patient is worse inside the house and better outside. He flares ten to thirty minutes after retiring as his mattress is the best generator of house dust. He is sensitive to cold air and is worse on damp days. This may also be a characteristic of food, and last he is worse with air conditioning.

One must rely upon the history in the food allergic patient to reveal the presence of the food sensitivity and to incriminate specific foods. The possible indications of food allergy are revealed in the post-prandial flare which occur immediately after eating and the periodic flares every afternoon at four thirty or the nocturnal attacks which awaken in the middle of the night. All food allergy is characterized by an absence of itching of the eyes, as a differential point in pollen and food. We must also remember that fatigue, insomnia and other toxic manifestations of allergy occur at lower levels of sensitization than the respiratory allergy.

Criteria

The criteria for specific food allergy, in analy-

zing the symptoms rest on several factors. For instance, does the food "lie heavy on the stomach"? (Can he eat pancakes?) Does the patient re-taste the food or is it returned undigested after three to seventy-two hours? The reactions of those specific days of the week or the period symptoms of certain times of the month must be elicited. Running all through these histories are certain important characteristics. One must realize simple inclusion of allergy in the differential diagnosis is easier and better than exclusion of all other diagnostic possibilities when these symptoms are discovered.

One of the most effective single measures in the PREVENTION of respiratory allergies is the elimination of unnecessary irritants to the respiratory tree. Smoking must be promptly stopped. One has not found asthma caused by this irritant but there are many who have no more trouble when the habit is discarded.

Tolerance to Foods

There are those individuals with "high inherant tolerance" and "low-inherant tolerance" for foods. The latter is most important. These individuals are recognized by their pecular complexion pallor resembling actual pastiness and "cafe au lait" coloring. Rinkel[5] feels that the failure to determine the inherant tolerance prior to application of restricted diets is encountered very frequently. This, more than any other single factor, accounts for the paradox of the most highly food allergic individual being regarded as non-food allergic simply because he did not show improvement from following a highly restricted diet. Briefly he breaks down to added foods and then, by Rinkel's cyclical concept, remains "masked" to the added food. No such patient should be kept on a highly restricted diet for more than seven to ten days. The keystone of all food allergic management is to have the patient symptom-free for five to seven days on his diet and then add foods.

Offending Allergen

An equaily effective factor in the MANAGEMENT of the allergy is removal of the offending allergen. In a whole family full of asthmatics it was discovered that the offending allergen was the pampas grass heads taken into the house and used decoratively throughout — in dining and living rooms as well as bed room. It required treatment difficulties for an individual's sore throat and finally a trip to the home. Most are not this simple. Another persistent bronchitis at Christmas cleared with removal of the tree.

One feels certain that the foods which are of greater incidence in the diet, that is, occurring most frequently in the diet, will likewise be the most plausible source for food allergy. If this be true, then corn, wheat, milk, egg, potato, orange, beans, beef, lettuce, tomato, coffee and pork will be the allergens most frequently incriminated in the total food list. The most significant thing in comprehending this "order of incidence" is the necessity for removing these food individually. Relief of symptomatology by removal of a food is not an important point in itself until one puts the food back in the diet and reproduces the same set of symptoms as before. This is the proof that the food in question is the allergen.

Earliest Signs

The earliest signs of respiratory food allergy— or a respiratory allergy for example other than typical hay fever and nasal congestion — are cough and clearing of the throat. The earliest indication of asthma is the wheezing caused by laughter and exercise.

Rinkel feels that the character of the sputum is important. It changes from frothy for a comparatively recent insult to purulent for an insult eighteen to twenty-four hours old. A brownish sputum is found with insults of greater age than forty-eight hours.

There are those individuals who feel that a pollen count is more significant than any other factor in the control of pollens and the other contributing causes to asthma. I feel that the weed study and the "weed hunt" is of prime importance in the early management of the respiratory allergic patient.

Rotation and Diversification

Another feature of significance and importance in the management of early preasthmatic or other

type of respiratory allergy is the institution of Rinkel's planned ROTATION and DIVERSIFICATION of diet. This simply introduces as many foods into the individual's diet as possible rather than limiting him to lamb, pears and rice with the addition of one new food daily, for example. This latter program ensures the breakdown of this individual for these foods with their constant re-use with the addition of new foods.

Rinkel diversifies as widely as possible. He adds as many foods to the diet as possible, depending on the individual means and foods are spaced as far apart as possible. Any number of foods are introduced at one feeding daily, one feeding every third or one feeding every fifth day. For instance, an individual taking orange juice today for breakfast would use four other juices before returning to orange juice if on a five day rotation. Beef can be rotated with lamb, poultry, pork, fish, rabbit, and even venison before returning to this possible allergen. These principles are carried through by careful preparation of monthly diet schedule.

Variance of Opinion

The multiplicity of preparations used for the treatment of asthma attest the variance of opinion as to cause. One of the earliest, simplest and yet most dangerous remedies was that used by Duke —ten grains of aspirin with two ounces of whiskey. Obviously we cannot give this to a child but the four minim dose of adrenalin is still a very efficacious emergency measure. Demerol is believed to be a dangerous drug in asthma. Most medicinal excipients are corn starch so these will make the corn-sensitive patient worse. ACTH is a substance being used with increasing frequency in all ages probably because of its prolonged stimulatory effect.

In closing, let me say that I as an otolarynologist, would not attempt the treatment of asthma without the close cooperation of one practitioner well versed in its medical management.

Pollen Control

Many allergists have noted that pollen control is considerably complicated by the injudicious use of steroids elsewhere. The skin-whealing response is altered to such a degree that titration becomes impossible and proper dosage equally so. Neither Rinkel nor Cowen will undertake treatment of a patient who has had steroids within the month and do not use them for treatment. Some of these asthmatics treated by this means may die rather unexpectedly. I know of nobody who has had personal experience with steroid insufflation as in Britain.

There are physicians who attempt patient management based on skin testing for pollen and foods, the cytoxic cell study, Gardner testing and, of late, the Godlowski regime or program. This last depends upon a sedated basal metabolic rate of minus fifteen regardless of PBI, radioactive I_2 uptake, serum cholesterols and all new and as yet undevised determinations of thyroid function. The study progresses on a concept of hypo-metabolism from there on.

Perhaps the patient with nocturnal food attacks should not eat an evening meal. His metabolism is slower and less active while asleep. There undoubtedly is less natural adrenalin secreted for relief and absorptive areas are contacted for greater periods of time.

We must all handle these problems when they are seen with our best efforts. None can ask more than this.

307 North Pennsylvania,

Roswell, N. M.

Bibliography

Management of Respiratory Allergy

1. James, J. Angell: A Laryngologist's View on Allergy Proc. Royal Soc. Med.; 50:10 827-837 October 1957
2. Hilsinger, Raymond L.: Allergic Headaches Trans. Am. Acad. Oph and Otol. 62:4 578-589 July-August 1958
3. Rinkel, Herbert J.: Post Graduate Lectures, University of Texas, Houston, Texas February 1957
4. Udenfriend, S.; Studies of Tryptophas and Serotonin in Patients with Malignant Carcinoid: Science: 123: 599 1956
5. Rinkel, H. J.; Randolph, Theron G.; Zeller, Michael: Food Allergy, C. C. Thomas, Springfield, 1951 pp 143-147
6. Rinkel, Herbert J.: Kansas City, Missouri : Personal Communication
   Cowen, D. Eugene: Denver, Colorado : Personal Communication

Palliative Treatment Of Benign Prostatic Hypertrophy

Value of Glycine-Alanine-Glutamic Acid Combination

By Henry M. Feinblatt, M.D.* and Julian C. Gant, M.D.**

In a controlled clinical investigation of benign prostatic hypertrophy, a course of medication with glycine-alanine-glutamic acid capsules† was found to be effective in reducing the size of the prostate and relieving the associated symptoms of discomfort, nocturia, delayed micturition, frequent urination and urgency. As compared with the controls, the results are statistically significant.

The value of the glycine-alanine-glutamic acid compound for relief of symptoms of benign prostatic hypertrophy was suggested by a chance observation made by one of us (J.C.G.)[1] and an associate.†† A group of allergic patients were being given an amino acid mixture and during the course of treatment one of the patients volunteered the information that all of his urinary symptoms had disappeared. This led to a trial of that particular amino acid compound in non-allergic patients with urinary symptoms. Patients with enlarged prostates and associated urinary symptoms experienced prompt and rather spectacular relief of their symptoms. A number of these patients have been observed over a prolonged period of time. They remain symptom free while taking the medication, but soon after discontinuing it the symptoms of urgency, nocturia, etc., returned. This has been the experience of several physicians who have tried the compound at our suggestion.

These original findings have been confirmed by an independent controlled clinical test.

Controlled Clinical Tests

To determine the value of the medication, a series of 40 cases of benign prostatic hypertrophy were treated with glycine-alanine-glutamic acid capsules for a period of three months. As controls, the same patients were given a placebo for a comparable period of two months. Our conclusions were drawn from this comparison.

The series included 40 men with diagnosed benign prostatic hypertrophy. Enlargement of the prostate was determined by rectal palpation. Patients having an indurated nodule located in one prostatic lobe were excluded because of possible malignancy (Jewett[2]). In addition, all subjects were given roentgenographic examination as an additional diagnostic precaution.

Additional Precaution

All patients were referred or checked by a urologist before assignment to this research program. Our consultations with urologists resulted in a decision in favor of conservative treatment. The conditions were diagnosed as benign and had not progressed at any significant rate of speed. If relief could be secured by non-surgical treatment, no advantage could be obtained by more radical procedures.

The age range was from 37 to 75, average 60. Weight ranged from 101 to 192 lb., average 163 lb. Height ranged from 65 to 73 in., average 68 in. The duration of the complaint ranged from one to six years, average four years.

The condition of the prostate on palpation was a swelling in nine cases, soft swelling in 23 cases, and prominent enlargement (easily felt) in eight cases.

The symptoms were discomfort in 35 cases, nocturia in 39, delayed micturition in 23, excessive frequency of urination in 29, and urgency of urination in 27 cases.

All patients with contributory diseases, as disclosed by the history and physical examination, were excluded from the group.

Placebo Cases

In the even numbered cases, as they presented themselves, the placebo capsules were substituted for glycine-alanine-glutamic acid capsules. They were given according to the same dosage schedule

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†Supplied as Prostall capsules by Metabolic Products Corp. of Boston, Mass. Each capsule contains 6¼ gr. of a mixture of glycine (amino-acetic acid), alanine and glutamic acid. The original formula was modified by addition of glycine by E. Y. Domina, B.A., a chemist, for definite biochemical reasons.

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Reprinted from the March 1958 issue of The Journal of The Maine Medical Association.

and the patients were not aware of the fact that they were taking a placebo. This regimen was continued for a period of two months.

The odd numbered cases, as they presented themselves, were given glycine-alanine-glutamic acid, two capsules three times daily after meals for two weeks, thereafter one capsule three times daily. This regimen was continued for a period of three months.

Later the placebo cases were switched to glycine-alanine-glutamic acid medication and vice versa. The results form a basis of comparison which gives a reliable indication of the therapeutic value of the medication.

All signs and symptoms were re-evaluated periodically in both groups. Routine chemical and microscopical examination of the urine was performed. Individuals with positive urinary findings were excluded from the group.

Results of Therapy

As shown in the table, the treatment was followed by reduction of prostatic enlargement and relief of the attending symptoms in significant percentages of cases. The size of the prostate was reduced in 92%, to normal size in 33%. Nocturia was relieved in 95% of cases (completely in 72%), urgent urination in 81%, frequency in 73%, discomfort in 71%, and delayed micturition in 70%. No comparable improvement was observed in the controls.

TABLE

COMPARISON OF THE THERAPEUTIC RESULTS

Glycine-Alanine-Glutamic Acid Capsules vs. Placebo Capsules

	Glycine-Alanine-Glutamic Acid	*Placebo*
Enlargement Reduced	92%	5%
Restored to Normal Size	33%	0%
Nocturia Relieved	95%	15%
Nocturia Relieved Completely	72%	5%
Urgent Urination Relieved	81%	11%
Frequency Urination reduced	73%	15%
Discomfort Relieved	71%	9%
Delayed Micturition Relieved	70%	4%

There were no adverse reactions in any case.

Rationale

During the course of medication we frequently observed disappearance of pitting edema in the legs and various parts of the body. Since the prostate and surrounding pelvic tissues are often congested and edematous in cases of benign prostatic hypertrophy, leading to pressure upon the base of the bladder in the region of the sensitive trigonum vesicae, the swelling leads to characteristic symptoms of bladder irritability which are associated with benign prostatic hypertrophy.

There is reason to believe that the anti-edemic action of glycine, alanine and glutamic acid reduces edematous swelling of the prostrate and neighboring pelvic tissues and in this way provides the therapeutic benefits described in this paper.

Edema is one of the most important symptoms of protein deficiency (Youmans[3]) and in such cases dietary protein and amino acids serve a useful function as a diuretic agent (Stare and Thorn[4]).

Glycine, alanine and glutamic acid are three of the dispensable amino acids (Elman[5]). As such they are unit components of food and tissue proteins. We must therefore consider the possibility of a definite metabolic effect in explanation of the results.

Therapeutic properties have been established definitely for certain individual amino acids. Thus methionine has proved to be useful in cases of cirrhosis of the liver (Beams and Endicott[6]), diaper rash (Goldstein[7]), and vitreous opacities (Eggers[8]). Administration of lysine to nutritionally substandard infants produces marked increases in body weight and appetite (Albanese et al.[9,10]). Many other examples of therapeutic effects produced by individual amino acids could be cited.

Glycine has been used with reported benefit in the treatment of various myopathies and peripheral vascular insufficiency. *(United States Dispensatory[11]).*

Favorable results with glutamic acid have been reported in the treatment of petit mal and psychomotor epilepsy (Price, Waelsch and Putnam[12]) and of mental deficiency (Zimmerman and Ross[13]; Albert, Hoch and Waelsch[14]). Numerous other therapeutic uses have been published *(United States Dispensatory[15]).*

We believe that ours is the first published report on the value of glycine, alanine and glutamic acid in the treatment of benign prostatic hyper-

trophy. It is hoped that other physicians will investigate this field of usefulness.

Discussion

Prostatic hypertrophy is part of the normal aging process and is undoubtedly present to a certain extent in all elderly men. After forty-five, more than 50% of all men develop benign prostatic hypertrophy. (Hinman[16]). Pathologically, the condition is a benign hyperplasia of the prostatic glandular tissue of an adenofibromatous nature.

Statistics supplied to the authors by the Metropolitan Life Insurance Company[17] report a death rate of 3.9 per 100,000 population from benign prostatic hypertrophy in 1953, 3.8 in 1954, and 3.7 in 1955.

As Chapman[18] has written, prostatic enlargement should be regarded, like arcus senilis and canities, as normal concomitants of old age which occur often enough to be regarded as a variety of the normal. Only when there is obstruction to the flow of urine does the condition become a disease. Usually the growth of the prostate slows down around the age of sixty. Non-progressive types of the disease should be treated expectantly. Chapman states, and we agree, that it is unjustifiable to remove the prostate merely because it is enlarged and has been associated with some urinary symptoms.

In the great majority of cases of benign prostatic hypertrophy, there is a large field for medical treatment including the use of glycine-alanine-glutamic acid mixture. Except in emergencies, conservative measures should be given a fair trial before resorting to prostatectomy.

Case Reports

Case 1. J.B., age 57, weight 181 lb., height 72 in., complained of prostatic symptoms dating back five years. He suffered from a feeling of pressure in the perineal region, discomfort on urination, difficulty in starting micturition, nocturia (twice per night), excessive frequency and urgency.

On rectal palpation enlargement of the prostate took the form of a soft swelling. Otherwise there were no physical findings. Roentgenographic examination was negative.

The diagnosis was benign prostatic hypertrophy.

Placebo capsules were administered according to a definite schedule: two capsules three times daily after meals for two weeks, thereafter one capsule three times daily. This regimen was continued for two months. The patient was not told that he was taking a placebo.

At periodic examinations and also at the end of the two months, there was no change in the size or condition of the prostate gland. None of the symptoms were relieved, even partially.

The patient was then switched to glycine-alanine-glutamic acid capsules, two capsules three times daily after meals for two weeks, thereafter one capsule three times daily. This regimen was continued for three months.

With this treatment the symptoms of discomfort, nocturia, difficulty in starting micturition, excessive frequency and urgency were relieved completely. On rectal palpation the size of the prostate gland was reduced to normal.

There were no untoward reactions and the urine was negative throughout both periods of observation.

Case 2

P.W., age 66, weight 152 lb., height 67 in., complained of prostatic symptoms dating back six years. He suffered from difficulty in starting micturition, scanty flow after beginning, discomfort on urination, a feeling of pressure in the perineal region, and nocturia (twice per night).

The prostate was palpated via the rectum and outlined as a soft swelling. Physical and roentgenographic examinations were otherwise negative.

The diagnosis was benign prostatic hypertrophy.

Placebo capsules were administered according to a definite schedule: two capsules three times daily after meals for two weeks, thereafter one capsule three times daily. This regimen was continued for two months. The patient was not informed that he was taking a placebo.

At each regular examination and also at the end of the observation period, no change was found in the size or condition of the prostate gland. None of the symptoms were relieved, even partially.

The patient was then switched to glycine-alanine-glutamic acid capsules, two capsules three times daily after meals for two weeks, thereafter one capsule three times daily. This regimen was continued for three months.

Under the glycine-alanine-glutamic acid treatment, the size of the prostate gland was reduced

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to normal. All of the symptoms of delayed micturition, scanty flow after starting, discomfort, perineal pressure and nocturia were relieved completely.

No untoward reactions were observed at any time and the urine remained constantly normal.

Conclusions

1. In a series of 40 cases of diagnosed benign prostatic hypertrophy, the use of glycine-alanine-glutamic acid capsules reduced the size of the enlarged prostate in 93% cases, including 33% in which the gland was restored to normal size. With the placebo only 5% showed partial reduction of prostatic enlargement, in no case to normal size.

2. The treatment relieved nocturia in 95% (complete relief in 72%), urgency in 81%, frequency in 73%, discomfort in 71%, and delayed micturition in 70%. With the placebo nocturia was relieved in 15% (complete relief in 5%), urgency in 11%, frequency in 15%, discomfort in 9%, and delayed micturition in 4%.

3. No untoward effects were observed in any case.

4. The modus operandi appears to be an antiedemic (diuretic) action whereby edematous swelling affecting the prostate gland and surrounding perineal tissues is reduced. A special metabolic activity may be a factor.

5. The great majority of cases of benign prostatic hypertrophy should be treated conservatively and the glycine-alanine-gluatamic acid mixture is recommended as an effective palliative.

References

1. Gant, J. C., jt. auth.
2. Jewett, H. J., Significance of the palpable prostatic nodule, *J.A.M.A.* 160:838, 1956.
3. Youmans, J. B., The clinical detection of protein deficiency, *J.A.M.A.* 128:439, 1945.
4. Stare, F. J., and Thorn, G. W., Protein nutrition in problems of medical interest, *J.A.M.A.*, 127:1120, 1945.
5. Elman, R., The practical use of amino acids in protein nutrition, *J.A.M.A.*, 128:659, 1945.
6. Beams, A. J., and Endicott, E. T., Histologic changes in livers of patients with cirrhosis treated with methionine, *Gastroenterlogy*, 9:718, 1947.
7. Goldstein, I. S., Napkin dermatitis caused by ammoniacal urine; treatment with methionine, *Arch. Pediat.* 66:553, 1949.
8. Eggers, H., Lipotropic substances for absorption of vitreous opacities, *New York J. Med.*, 51:2255, 1951.
9. Albanese, A. A. et al., Biochemical and nutritional effects of Lysne-reinforced diets. *Am. J. Clin. Nutrition*, 3:121, 1955.
10. Albanese, A. A. et al., Lysine supplementation in infant feeding; dosage considerations, *New York State J. Med.*, 55:3453, 1955.
11. *U. S. Dispensatory*, ed. 25, Philadelphia, J. B. Lippincott Co., 1955, pp. 63-64.
12. Price, J. C., Waelsch, H., and Putnam, T. J. dl-Glutamic acid hydrochloride in treatment of petit mal and psychomotor seizures, *J.A.M.A.*, 122:1153, 1943.
13. Zimmerman, F. T., and Ross, E., Effect of glutamic acid and other amino acids on maze learning in white rat, *Arch. Neurol. & Psychiat.*, 51:446, 1944.
14. Albert, K., Hoch, P., and Waelsch, H., Preliminary report on effect of glutamic acid administration in mentally retarded subjects, *J. Nerv. & Ment. Dis.*, 104:263, 1946.
15. Ibid. ref. No. 10, p. 610.
16. Hinman, F., The obstructive prostate, *J.A.M.A.*, 135:176, 1947.
17. Metropolitan Life Insurance Company, National Office of Vital Statistics, Special Reports Volume 46, No. 1.
18. Chapman, T. L.,, Expectant treatment of benign prostatic enlargement, *Lancet*, 2:684, 1949.

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Prevention of Pulmonary Hyaline Membrane Results of a Study

By Jack Curry Redman, M.D., A.A.G.P., Albuquerque

In December, 1957, the author (1) suggested an iatrogenic factor in the development of hyaline membrane disease, namely the improper positioning of the susceptible newborn infant. It was suggested that placing these infants in horizontal or head-down position imposes an additional burden on their marginally-adjusted cardio-vascular and respiratory systems. It was recommended that all susceptible newborns (prematures and those delivered by Cesarean section) be placed immediately after birth in the head-and-body elevated position, with humidification of some type, and oxygen only for severe cyanosis.

Since publication of the preliminary report the author has completed a six-month study, the results of which add support to the suggested plan for the prevention of hyaline membrane disease. Between October 1, 1957, and April 1, 1958, information cards were filled out by the nurses of two hospitals' newborn nurseries in this city on every baby whose birth weight was less than five pounds eight ounces and on every baby delivered by Cesarean section. These cards, when completed, provided each baby's name, sex, weight, route of delivery; its position in the incubator (flat, elevated, or head-down); the presence or absence of cyanosis, grunting, or retraction; the type of humidification; the use of oxygen; the presence or absence of hyaline membrane disease.

The series presented contained 93 vaginal deliveries and 36 Cesarean section deliveries (Figure 1.). Of the vaginal deliveries 62 babies were placed flat in their incubators and 30 were elevated ten to twenty degrees; one was placed head-down. Of the section group 16 babies were placed flat and 18 were elevated; two were placed head-down. There were 20 deaths from all causes in the group delivered vaginally (Figure 2.). There were five deaths from all causes in those delivered by Cesarean section (Figure 3.).

There were nine cases of hyaline membrane disease in the vaginal group, for an over-all incidence

VAGINAL DELIVERIES

Position	Number	Hyaline Membrane	Deaths (Total)
Flat	62	7*(11%)	16 (24%)
Elevated	30	2 (7%)	4 (13%)
Down	1	0 (0%)	0 (0%)
Total	93	9 (10%)	20 (22%)

*One Baby Recovered

SECTION DELIVERIES

Position	Number	Hyaline Membrane	Deaths (Total)
Flat	16	3 (19%)	3 (19%)
Elevated	18	0 (0%)	2 (11%)
Down	2	0 (0%)	0 (0%)
Total	36	3 (8%)	5 (14%)

Figure 1

VAGINAL DELIVERIES

FLAT GROUP

Weight	Cause of Death
(1) 3# 2 oz.	Hyaline Membrane (Autopsy)
(2) 2# 1 oz.	Hyaline Membrane (Clinical Diagnosis)
(3) 4# 2 oz.	Congenital Heart Disease (Clinical Diagnosis)
(4) 3# 12 oz.	Resorptive Atelectasis (Autopsy)
(5) 4# 4 oz.	Hyaline Membrane & Resorptive Atelectasis (Autopsy)
(6) 2# 7½ oz.	Prematurity (Clinical Diagnosis)
(7) 2# 15 oz.	Prematurity (Clinical Diagnosis)
(8) 4# 4 oz.	Hyaline Membrane (Autopsy)
(9) 5# 4 oz.	Hyaline Membrane (Autopsy)
(10) 2# 1 oz.	Resorptive Atelectasis (Autopsy)
(11) 5#	Internal Hydrocephalus (Autopsy)
(12) 5# 1 oz.	Aspirated Amniotic Fluid (Autopsy)
(13) 3# 9 oz.	Pulmonary Congestion & Hemorrhage (Autopsy)
(14) 3# ½ oz.	Cerebral Hemorrhage & Prematurity (Autopsy)
(15) 5# 3 oz.	Hyaline Membrane (Autopsy)
(16) 4# 1 oz.	Unexpanded Lungs (Clinical Diagnosis)

ELEVATED GROUP

Weight	Cause of Death
(1) 2# 15 oz.	Duodenal Atresia (Post-Operative)
(2) 2# 2 oz.	Unexpanded Lungs (Clinical Diagnosis)
(3) 2# 11 oz.	Hyaline Membrane (Autopsy)*
(4) 2# 8 oz.	Hyaline Membrane (Autopsy)*

*Twins

Figure 2

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SECTION DELIVERIES

FLAT GROUP

Weight	Cause of Death
(1) 5# 13½ oz.	Hyaline Membrane (Clinical Diagnosis)
(2) 6# 8 oz.	Hyaline Membrane (Autopsy)
(3) 5#	Hyaline Membrane (Autopsy)

ELEVATED GROUPS

Weight	Cause of Death
(1) 4# 13½ oz.	Atelectasis (Autopsy)
(2) 3# 7 oz.	Atelectasis (Autopsy)

Figure 3

of ten per cent. Seven of these cases were in the "flat group" six died and one recovered. There were two cases (a set of twins), both of whom died, in the "elevated group". There were three cases of hyaline membrane disease in the section group, for an over-all incidence of eight per cent. All of these babies were in the "flat group", and all of them died.

Half of all the babies in the two groups received additional humidification by means of a compressed air-distilled water nebulizer apparatus. It is interesting that all of the babies who developed hyaline membrane disease received the "mist treatment". Conversely it is interesting that among the babies who did *not* receive the "mist treatment" there was not a case of hyaline membrane disease or of resorptive atelectasis. The use of wetting agents apparently had little effect on the incidence of hyaline membrane. Oxygen, usually at two liters per minute, was given to a number of babies without appreciable harm. All of the babies who died received oxygen for cyanosis as it developed.

DISCUSSION

The incidence of hyaline membrane disease has increased gradually during the past few years. Paradoxically, it has increased in spite of our "better care for prematures". The same paradox existed not too long ago in the case of retrolental fibroplasia. At long last we realized that one of our concepts of proper premature-infant care had to be changed. Once changed, retrolental fibroplasia stopped adding its terrible number of blind babies to the world. It seems to this observer that another change in concept is due, and that, once the change is made, the disease we know as hyaline membrane will cease to exact its toll.

As stated before by many authors, the best preventative measure against hyaline membrane disease is the cessation of premature and Cesarean section deliveries. Inasmuch as we are currently unable to achieve such a cessation, we must, of necessity, concern ourselves with better techniques of newborn care.

Significant Finding

One of the more significant physical findings in babies delivered prematurely or by Cesarean section is the decrease in or even absence of normal vesicular breath sounds, especially over the bases, immediately after birth. Within the first few hours of life the breath sounds may be heard to appear gradually. During these first few hours these babies' cardio-vascular and respiratory systems are attempting to adapt to an outside environment into which the babies have been suddenly or prematurely admitted. It is during these first few hours that we have seen hyaline membrane develop all too frequently.

Our clinging to custom is never-changing. Custom has dictated that all newborns should be placed flat or head-down, ostensibly for "postural drainage". These positions actually increase the chance of vomiting by virtue of the pressure of the abdominal viscera against the stomach. Further, these positions increase the pressure against the diaphragms, and thereby limit their movement by the same gravitational mechanism. It would seem that such positions place our babies, especially prematures, in double jeopardy. This observer suggests that we abandon the customary non-physiologic positioning of our newborns, and utilize, rather than fight, the force of gravity. I am sure that Sir Issac Newton, himself a small premature at birth, would concur.

Significant Analogy

One has only to picture an asthmatic patient, or one with pneumonia or congestive heart failure lying flat or head-down in bed to see a very thought-provoking analogy. Certainly our knowledge of normal and abnormal respiratory physiology should suggest that the position of choice for a newborn baby, especially one delivered prematurely or by Cesarean section, is in the head-and-body-elevated position. These babies should be placed in this position even as the cord is being tied, and they should remain in this position until they have adapted to the outside environment. The lowering to horizontal should be a gradual procedure.

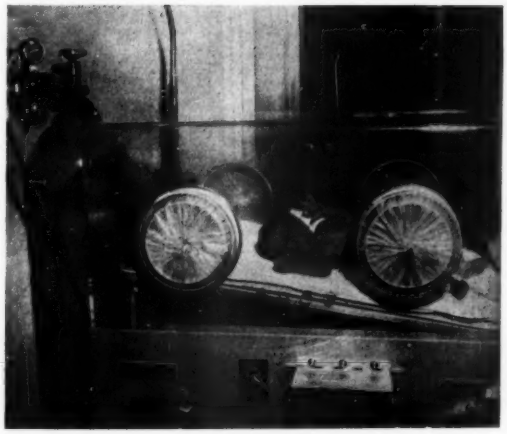

Figure 4
The Suggested Newborn Position is Shown

Supersaturated environments ("mist treatment") probably contribute to further respiratory distress in some cases. A humidity of 85-90% seems to be quite adequate in most cases. The advantage of the use of wetting agents has not been striking. Obviously a great many variables exist in each case. Much closer harmony between the obstetrician and the pediatrician must be developed before a uniformity of treatment may be realized.

CONCLUSIONS.

Hyaline membrane disease of the newborn is felt to represent the end-result of respiratory and cardio-vascular insufficiency. Except for prematurity and delivery by Cesarean section, probably the greatest factor in development of this disease has been iatrogenic. The flat or head-down newborn position is not physiologic and should be abandoned in favor of the head-elevated position.

Susceptible babies should be placed in the elevated position immediately after birth, even as the cord is being tied. They should be placed in incubators in an humidified atmosphere of 85-90%. Super-saturated atmosphere ("mist treatment") should be avoided when possible. The degree of elevation should be, ideally, 10 to 20 degrees (Figure 4.). The use of oxygen should be reserved for the treatment of severe cyanosis. The use of wetting agents, antibiotics, and indicated medication must be individualized.

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ACKNOWLEDGEMENT.

The author expresses his gratitude to the newborn nursery personnel of the Presbyterian Hospital Center and St. Joseph Hospital for their splendid cooperation which made this study possible. The author is also indebted to his father, C. E. Redman, for the photo (Figure 4.).

Reference

1. Redman, J. C.; The Role of Newborn Position in the Development of Pulmonary Hyaline Membrane; Southwestern Medicine, December 1957.

Abstract

"Therapeutic Notes", published by Parke, Davis & Company contains an illuminating article entitled "Problems of the Newborn". Of paramount interest is the problem of the premature infant. The multiple factors playing a role in premature labor and delivery (fetal anomalies, maternal accidents, blood diseases, multiple pregnancy, hemorrhage, infection, environment) are discussed. Preventive measures suggested are: better living conditions in underprivileged groups; earlier, more efficient, and continuous prenatal care; improved obstetric management; and greater availability of skilled nursing and medical care.

Special problems of the premature (cyanosis, anemia, infection, surgical conditions) infant are discussed, along with preventive and therapeutic measures. Pulmonary hyaline membrane is discussed. Its frequent occurence among prematures, babies delivered by Cesarean Section, and babies delivered of diabetic mothers is stressed. The article states that there are "no specific preventive measures". An article by Dr. Redman in this issue of Southwestern Medicine is concerned with this subject.

"Whatever the problem of the Newborn", the Parke, Davis article concludes, "it is recognized increasingly that present knowledge, widely and intensively applied, can bring about significant reduction in loss of infant life".

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Iodo-Niacin In Treatment of Sinusitis, Bronchitis and Otitis

By James N. Adams, M.D., Longview, Texas

In a group of 30 case studies of sinusitis, bronchitis and otitis media or interna, Iodo-Niacin* medication was employed orally and parenterally with highly satisfactory results. No iodism or adverse reactions were observed in any case.

Iodo-Niacin tablets contain potassium iodide 135 mg. and niacinamide hydroiodide 25 mg. The usual dose is two tablets three times a day.

Iodo-Niacin ampuls 5 cc. contain niacinamide hydroiodide 50 mg., sodium iodide 20 mg., niacinamide 20 mg., dextrose five per cent, chlorobutanol (preservative) 0.45 per cent, and water for parenterals q.s. ad five cc. The usual dose is five cc. twice weekly by intramuscular injection.

For over a hundred years the iodides have been used extensively in the treatment of a wide variety of diseases, especially arteriosclerosis, syphilis and goiter. However, physicians have been discouraged by one serious disadvantage: namely, iodism occurring in a large percentage of cases when the treatment is continued in full dosage sufficiently long to obtain satisfactory therapeutic results.

Prevention of Iodism

In 1955 Feinblatt, Feinblatt and Ferguson[1] reported on the effectiveness of combining niacinamide with potassium or sodium iodide as a means of preventing iodism even on long continued medication in full dosage. They administered full doses of Iodo-Niacin to 59 patients with arteriosclerosis, for a year or longer, with marked therapeutic benefit and not a single case of iodism. Niacinamide was found to prevent iodism as effectively as pellagra, a condition with which iodism is closely related etiologically.

In a later study the same authors[2] treated 50 cases of arteriosclerosis with intramuscular injections of Iodo-Niacin. There was improvement in every case and not a single instance of iodism. Significant relief of the following symptoms was observed: vertigo, depression, disorientation, excessive fatigue, vague abdominal distress, headache, emotional instability and anorexia.

Abrahamson and Abrahamson[3] in 1956 reported a group of 22 cases of retinal and vitreous hemorrhages and 89 of vitreous floaters which were treated with Iodo-Niacin tablets. Compared with a control group on placebo medication, the treated patients showed much more rapid and complete absorption of retinal and vitreous hemorrhages and also improvement in vitreous floaters. No case of severe iodism occurred with the use of Iodo-Niacin.

Therapeutic Uses of Iodides

An iodide medication which can be administered indefinitely in full dosage without fear of oidism has obvious advantages. It is indicated for a wide field of therapeutic purposes in addition to the treatment of sinusitis, bronchitis, otitis media and interna, and Meniere's syndrome.

In a discussion of the expectorant action of the iodides, Sollmann[4] in 1957 wrote: "Potassium iodide increases the secretion of bronchial mucus and renders it less viscid; it is therefore used in cough and especially in chronic asthma."

Carryer et al.[5] in 1946 discussed the use of iodides as an expectorant for asthmatic patients: "The mucus in the bronchioles of an asthmatic patient is usually viscous and tenacious. Much exhausting coughing on the part of the patient is necessary to dislodge and raise this mucus. Therefore an expectorant is of utmost value to the patient, particularly when bronchitis is a prominent etiologic factor. Iodides in liberal quantities are the most useful of the expectorants."

Iodine, internally administered, is very rapidly absorbed by all mucous membranes (Foote[6]). In a series of cases of common cold in which iodine was used orally, the majority of patients showed diminished turgescence of the nasal mucosa and relief of dryness of the nose and throat within half an hour.

*Iodo-Niacin tablets and ampuls were supplied for this investigation by Cole Chemical Co., of St. Louis, Mo.

Kovnat[7] recommended iodine orally as an abortive for the common cold. He said that, if iodine is given at the beginning of a cold, the early symptoms are relieved and the second stage of congestion and mucoid discharge may be prevented.

Discussing the action of iodides on the respiratory organs, Bastedo[8] stated that "there is increased fluidity and increased flow of mucus in nose, throat, and bronchi, associated with an affinity of iodine for the respiratory epithelium."

Clinical observations by Jarvis[9] demonstrated "that iodine taken into the body reduces the size of the lymphoid tissue in the upper respiratory tract." He noted that oral iodine relieved nasal obstruction and decreased the size of the turbinates and the lymphoid nodules on the posterior pharyngeal wall.

Clinical Data

The group included 30 case studies on 20 patients, seventeen females and three males. The age range was from 20 to 65, average 44. The diagnosis was sinusitis in 16 cases, otitis media or interna in seven cases, bronchitis in four cases, and Meniere's syndrome in three cases. In four cases sinusitis was complicated by bronchitis and in three cases by otitis. Meniere's syndrome was a complication in three cases.

The principal complaints included pain, headaches, nasal congestion, postnasal drip, tinnitus, vertigo, symptoms of eustachian tube blockage, sore throat, cough and expectoration.

The basic treatment consisted of Iodo-Niacin, two tablets three times a day, supplemented by intramuscular injections of Iodo-Niacin once or twice a week. In some cases penicillin and other antibiotics were also used as indicated.

There was improvement in all 16 cases of sinusitis treated with Iodo-Niacin and in three cases the condition was cleared up. All seven cases of otitis showed improvement, including clearing of the symptoms in a case of Meniere's syndrome. Cough and expectoration were relieved in the four cases of bronchitis.

No iodism or adverse reactions were observed in any case under treatment with Iodo-Niacin.

Discussion

My experience confirms the value of iodide therapy for sinusitis and bronchitis and also suggests its use in suitable cases of otitis media and interna including Meniere's syndrome.

Iodo-Niacin injections worked most satisfactorily in severe infected cases of sinusitis and bronchitis rather than in the allergic types. Both the tablets and ampuls were very helpful in loosening the bronchial secretions and as an expectorant. Iodo-Niacin also had a non-specific effect on acute pansinusitis of the infectious type.

In the allergic type of sinusitis results with Iodo-Niacin were encouraging but questionable. In some cases the use of Iodo-Niacin was combined with antibiotics, such as penicillin and tetracycline. In such cases it may be difficult to evaluate properly which drug helped the patient.

After evaluation of all of the clinical data, it is my opinion that Iodo-Niacin is very helpful in the chronic infected types of both sinusitis and bronchitis.

The complete absence of iodism and adverse reactions in patients treated with Iodo-Niacin confirms the previous reports of Feinblatt, Feinblatt and Ferguson[1,2] and of Abrahamson and Abrahamson[3].

Conclusions

1. A clinical investigation showed Iodo-Niacin tablets and ampuls to be very helpful in the treatment of severe infected cases of sinusitis and bronchitis.
2. The cough and expectoration of bronchitis were effectively relieved by Iodo-Niacin.
3. Favorable results in a small group suggest the value of Iodo-Niacin in the therapy of otitis media, otitis interna and Meniere's disease.
4. No iodism or untoward reaction to Iodo-Niacin was observed in any case.

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References

1. Feinblatt, T. M., Feinblatt, H. M., and Ferguson, E. A., Jr., Treatment of Arteriosclerosis and vague abdominal distress with niacinamide hydroiodide (without side-effects), *Am. J. Digest. Dis.* 22:5, 1955.
2. Feinblatt, T. M., Feinblatt, H. M., and Ferguson, E. A., Jr., Arteriosclerotic syndromes, niacinamide hydroiodide (parenteral) treatment, *M. Times* 84:741, 1956.
3. Abrahamson, I. A., Jr., and Abrahamson, I. A., Sr., Use of Iodo-Niacin for retinal or vitreous hemorrhages and vitreous floaters, *Am. J. Ophth.* 42:771, 1956.
4. Sollmann, T., *Manual of Pharmacology*, Philadelphia, W. B. Saunders Co., 8th ed., 1957, p. 1121.
5. Carryer, H. M., Prickman, L. E., Maytum, C. K., and Koelsche, G. A., Treatment of a seriously ill asthmatic patient, *J.A.M.A.* 131:21, 1946.
6. Foote, R. R., Plea for more general use of iodine in treatment of common cold, *M. Press* 209:335, 1943.
7. Kovnat, M., Abortive for common cold, *M. Rec.* 153:175, 1941
8. Bastedo, W. A., *Pharmacology, Therapeutics and Prescription Writing*, Philadelphia, W. B. Saunders Co., 5th ed., 1947, p. 723.
9. Jarvis, D. C., Lymphoid tissue in upper respiratory tract, *Laryngoscope* 52:188, 1942.

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APHORISMS and MEMORABILIA

Miscellaneous Truths and Concepts

(Continued)

11. "The following liver function tests, in the order of their importance, are the ones which are used routinely in our clinic to determine the operability of these patients: (Portal Cirrhosis) 1. the serum albumin, 2. the presence of ascites and whether or not it fails to respond to medical therapy, 3. the cephalin flocculation test, 4. the prothrombin time, 5. the serum bilirubin level, and 6. the bromsulphalein retention test. The serum albumin level is considered the most significant test in determining the operability of the patient."—LINTON, *Amer. Journal of Med.*, June 1958, p. 944.

12. ". . . it is our rule now that the serum albumin level must be above three gm. per cent in all patients who undergo shunt surgery. If it is not, appropriate measures are instituted to elevate it, including the intravenous administration of human serum albumin concentrate." LINTON, *Amer. Journal of Med.*, June 1958, page 944.

13. ". . . in the majority of patients who are grossly icteric surgery is usually deferred until the jaundice clears." — LINTON, *Amer. Journal of Med.*, June 1958, page 944.

Ageing

14. "The speed which people grow old depends as much on social factors as it does on purely physical processes. Ageing is quickened by such things as the loss of interests due to compulsory retirement or frailty; loss of relatives and friends due to the inevitable result of living longer; loss of financial security due to a decline in income or in the value of money; loss of emotional security due to changing environment; loss of status through ceasing to be primarily a contributor to society; and loneliness. Singly or collectively, these are responsible for much of the frustration, disappointment, anxiety, depression and unusual behaviour amongst elderly citizens."—*British Medical Journal*, June 21, 1958, page 1457.

15. "The ultimate value of culture is to suggest standards of good and evil which science alone cannot supply, and this should be remembered in all our study of culture in the past and in the present." — *Understanding History*, BERTRAND RUSSELL, p. 41, 1957. Wisdom Library.

16. "There are three things to be considered about an organization: what it offers to the public, what it offers to its own rank and file, and what it offers to its leaders. The last of these too often, in practice, outweighs the other two."—RUSSEL, *loc. cit.*, page 50.

Pollen Count

17. "The highest pollen count in almost every area occurs between seven and nine-thirty in the morning. There is no reason at all to let the patient go around from the time he arises until after breakfast snuffing and snorting like a fire engine. Give him a capsule (for hay fever) immediately upon arising."—DR. PAUL WILLIAMSON, *American Practitioner*, July 1958, page 1073.

18. "There are four categories of causes of secondary polycythemia in cardiopulmonary disease and with the use of a simple vital capacity, timed vital capacity, maximal breathing capacity and arterial blood studies under various conditions one can differentiate these four causes fairly easily so long as they are pure uncomplicated examples at the time of study."—DR. JOHN H. KNOWLES, *Amer. Practitioner*, June, 1958, page 978.

19. "A single inspiratory chest x ray is notoriously inadequate in the diagnosis of early obstructive emphysema."—*Loc. cit.*, page 979.

20. "When an electrocardiogram, particularly one taken as a part of a general examination, shows complete right branch block, or either complete or incomplete left branch block, the patient should have a more searching cardiac examination, including a careful history and physical examination of the heart, than would otherwise be done. If these studies fail to reveal any findings pointing to heart disease, the patient should not be told that he (or she) has cardiac abnormality, nor should his life or activities be altered in any way."—DR. F. D. JOHNSTON, *Modern Concepts of Cardiovascular Disease*, July, 1958, page 471.

MONTHLY CLINICAL PATHOLOGICAL CONFERENCE
EL PASO GENERAL HOSPITAL

NOVEMBER 20, 1958

FREDERICK P. BORNSTEIN, M. D., *Editor—Case No.* 1106
Presentation of case by J. Edward Stern, M.D.

History - Nathan Kleban, M.D.

An 11 month old Latin-American boy from Dona Ana County, New Mexico, was admitted to the hospital on Aug. 13 and died Aug. 15, 1958.

The infant's general health had been fair. He had diarrhea and fever for one week during the middle of July. A polio immunization injection was administered on Aug. 1. Irritability, fever, vomiting and diarrhea began on Aug. 9. Respiratory distress and weakness of the left leg were observed on the day of admission. The patient's doctor referred him to the hospital, reporting that the spinal fluid contained 13 cells, 3 of which were lymphocytes, and 95 mg. % protein.

An only sibling was said to have a cold.

Physical Examination:

On admission the patient was in coma, responded to painful stimulus. Respirations were shallow, irregular, 38 per minute. Temperature was 104, pulse 120. He appeared flushed, normal in development and state of nutrition. Anterior fontanel was two cm., soft. Neck and back were supple. Brudzinski and Kernig signs were absent. Abdominal and cremasteric reflexes were not elicited.

Deep tendon reflexes of the right lower extremity were more active than the left. Gag reflex was present. Extremities were somewhat resistant to flexion. Plantar reflexes were extensor bilaterally. Tissue turgor was decreased slightly. Heart rhythm was equivocally gallop. There were no other remarkable physical findings.

Laboratory Results:

There were 14,000 WBC; 64 segs., 36 lymphs. Hematocrit was 32, hemoglobin 10.8. Urine was pale yellow, turbid, alkaline, 1+ albumin, no sugar, 0-3 WBC and 0-1 fine granular casts/HPF. Blood sugar was 133, blood urea nitrogen 9.2 mg. %. Tuberculin skin test was negative at 48 hours. Spinal fluid pressure was 'normal', color a "little yellowish', contained many RBC; 2 lymphocytes/cu. mm.; sugar 130 and protein 124 mg.%; chloride 125 mEq/L. Blood was drawn to be sent to New Mexico State Health Department for viral studies.

Hospital Course:

There were five loose stools. Parenteral fluids, choranphenicol and acetelsalicylic acid were prescribed. Cyanosis appeared and improved only slightly with oxygen. Temperature dropped to 101.

On the following day there were a weak corneal reflex, hyperactive deep tendon reflexes and stiffness of back and legs without paralysis. Respirations improved after pharyngeal aspiration. Temperature rose to 106 followed by twitching movements of the left side of the face and left hand.

Temperature was brought down to 101.6. A gastric tube was inserted. Twitching movements of the left side of the face recurred. Myoclonic movements of the right ankle and foot lasting 30-40 seconds with an interval of several minutes persisted until death. Opisthotonus was noted. Temperature rose to 104. Cheyne-Stokes respirations decreased to two per minute. Heart rate slowed from 200 per minute then stopped while artificial respiration was being given. Death occurred 54 hours after admission.

Dr. Stern:

When I first looked over the clinical summary of this case it did not impress me as being particularly involved. One is dealing with a small child with fever, prostration and symptoms which are very suggestive of a cerebral rigidity. The child comes from a county of New Mexico which we know has had a good deal of encephalitis during the latter part of the summer, either of the St. Louis or equine type.

However, on going over the case more carefully, it struck me as being much more interesting than I had first thought and I began to analyze it a good deal more closely. We are dealing with, as you already know, an 11 month old Latin-American boy from Dona Ana County who was

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admitted on August 13 and died August 15, 1958. The infant's general health had been fair. He had diarrhea and fever for one week during the middle of July. What relevance this has to the later developments, I don't know.

A polio immunization was administered on Aug. 1, 12 or 13 days before the child was admitted to the hospital. The relationship of the polio immunization to the later clinical picture I think cannot be stated with certainty, although it may be a very important point in the history.

Irritability, fever, vomiting and diarrhea began on Aug. 9, nine days after the injection. Respiratory distress and weakness of the left leg were observed on the day of admission. The patient's doctor referred him to the hospital, reporting that the spinal fluid contained 13 cells, three of which were lymphocytes and an elevated protein.

Fever, pleocytosis by which one generally means a cell count of over 10 cells, and an increase in cerebro-spinal fluid protein, make one think mostly of the inflammatory diseases of the central nervous system. On admission the patient was in coma, responded to painful stimuli, respirations were embarrassed, shallow and 38 per minute.

Temperature was 104, pulse 120. He appeared flushed. Development and nutrition were normal. This observation is important in the light of what I am going to say later. The anterior fontanel was two cm. and soft, the neck and back were supple, Brudzinski and Kernig signs were absent.

The abdominal and cremasteric reflexes were not elicited, suggesting the possibility, at least in the case of the abdominal reflexes, that pyramidal tracts were involved, either by direct infection or by compression.

The deep tendon reflexes in the right lower extremity were more active than the left, gag reflex was present, the extremities were somewhat presistant to flexion. I take this to mean probably there was already some degree of rigidity due to decerebration.

The plantar reflexes were extensor bilaterally. Now I know that there is an old and well established teaching in medicine, pediatrics and neurology, that extensior plantar responses in small children are not significant. I am not sure that this statement is always correct.

I think that the extensor plantar response in a small child, specially if unilateral, may be very significant. Significant of what? — Of pyramidal involvement. In this case the extensor responses occur bilaterally, they could represent—I would state this with certainty—part of the whole syndrome of decerebration.

Leukocytosis

There was a leukocytosis, with 65% segs, hematocrit was 32, Hb. 10, which may be a little low although the pediatricians may feel that this is within normal range. The urine contained 1+ albumin, probably associated with the fever, no sugar, a few leukocytes, an occasional granular cast. The blood sugar was not elevated, it was 133, the urea nitrogen was 9.5, mg. per 100 cc., not elevated, the tuberculin skin test was negative at 48 hours.

I am glad to know that this was done, even though with an overwhelming general tuberculous infection of course the skin test could be negative. Spinal fluid pressure was thought to be normal, the color a little yellowish, there were many RBC,2 lymphocytes/cu. mm., sugar 130 and protein 124 mg.%, chloride 125 mEq/L. The spinal fluid sugar, (I hadn't really noted this before) which ordinarily has a value half that of the blood sugar is high, but perhaps the child was receiving glucose at the time this determination was made. Blood was drawn to be sent to New Mexico State Health Department for viral studies.

In a situation of this type even though it may seem at the outset a very simple one, there are a number of examinations which can usually be made rapidly and without any special risk to the child.

Careful examination of the skin for infection, eruption or tick bite; examination of the eyes for strabismus or inequality of the pupils; examination of the ears for otitic infection, mastoiditis, which might lead one to suspect either a primary jugular thrombosis or brain abscess, either of the temporal lobe or the cerebellar hemisphere; examination of the nose for purulent secretion, lung X-ray to look for abscess or miliary lesions although unlikely in a child well nourished; X-ray examination of the skull to look for evidences of pressure, mastoiditis, sinusitis, in other words infection in one of the air-spaces of the skull, and for scattered calcifications.

What about electroencephalograms in a situation like this? According to some statistics which are available, 30 per cent of patients with acute encephalitis have normal electroencephalograms, and of course abnormality of the electroencephalogram would depend on the severity of the reaction. If there were not enough compression, edema, interference with circulation to embarrass dendrites,

which are thought to be the source of the potentials of the electroencephalogram you could easily infer that the electroencephalogram would be normal.

In some, one is very likely to see large, slow stupor waves derived from all leads. These would not be of any particular diagnostic value, they would tell you, what you already know from inspection, that the patient is comatose.

In encephalitis, if the record is positive, the electroencephalogram may show either focal or diffuse slow activity going down to seven cycles per second, three cycles per second or even slower, depending on the portion of the brain which bears the brunt of the infection, and encephalitis is not always a uniformly distributed process.

Brain Abscess

In brain abscess you may find, barring the effects of overwhelming stupor a very distinctive electroencephalogram, with focally very large, very slow waves.

Without any more ado about this, in the absence of any supporting data for another diagnosis, and in the absence of contradictory data, I would simply go along for the moment with the diagnosis suggested at the onset, of a St. Louis or a Western type of encephalitis.

The prognosis of encephalitis, no matter how mild it may appear to be at the onset, is always very doubtful, for the reason that the treatment is always very unsatisfactory. I will come back to treatment a little later.

In the cerebro-spinal fluid in encephalitis or polioencephalitis, one may or may not find xanthochromia, red blood cells, normal or elevated white cell count, and normal or elevated protein, although I think that the protein would usually be elevated even if there were no other abnormalities. In poliomyelitis the spinal fluid may be absolutely non-contributory. The case clinically may be obviously one of polio but the spinal fluid may be normal.

In obscure cases I think it is important to get a Kahn test to try quickly to exclude syphilis. In the old days we used to say that in tuberculosis meningitis the chloride in the cerebro-spinal fluid was depressed and at one time the Levinson test was popular.

Meningeal Irritation

What about the lack of signs of meningeal irritation? I think what one is observing here rather than signs of meningeal irritation are rather those of decerebration. In a prostrated individual, I think it may be entirely possible to observe clinically that there are no signs of meningeal irritation. Now in addition to viral blood studies, I think it may be advisable to this area routinely to obtain agglutination test for OX19.

In looking over my old notes of encephalitis I found that I had prepared a list a long time ago, and in the light of the experiences, many of them unfortunate in recent years, I have had many occasions to amplify the list. Without mentioning everything, I will touch on some of the causes rapidly: Heavy metal poisoning such as arsenic, which we used to see in the old days with syphilitic treatment and lead are important; also methyl and ethyl alcohol poisoning, the latter probably more on the basis of an avitaminosis than anything else.

The best known effect of alcohol is Wernicke's acute superior hemorrhagic polioencephalitis in which the mid-brain, the part where the occular motor nuclei and red nucleus are located, bears the brunt of the disease.

Of course alcohol produces many other effects on the central and also on the peripheral nerve system. Toxic and allergic causes of encephalitis include measles, mumps, varicella and rabies innoculation. This is the so-called perivenous encephalomyelitis and I think is usually accompanied by demyelinization around cuffed veins.

Viral infections, rabies of course is a very well known one and we have had some examples of that here in the last couple of years. Rabies of course is characterized by intracytoplasmic inclusions. There is another type of inclusion encephalitis in which the inclusion bodies are intra-nuclear.

Under viral infections I think one can mention the Guillain-Barre syndrome, poliomyelitis, (which should always be thought of really as a polioencephalitis), encephalitis lethargica which is the type A encephalitis, and then all the other encephalitides of related group, the type B, including the Japanese, St. Louis, Western and so on. Herpes encephalitis is still another one of the viral type.

There are many rickettsial infections which invade the central nervous system, including typhus, Brill's disease or endemic typhus, Rocky Mountain spotted fever and scrub typhus. There are all sorts of bacterial infections, acute and chronic.

Meningococcus meningitis is strictly speaking a meningoencephalitis, as are the meningo-encephalitides of the embolic varities derived from organ-

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isms on the heart valves and mural endocardium and lung.

Leptospirosis, Weil's disease and a couple of other very rare ones like pretibial fever, then the spirochetal infections are really quite important, or at least used to be. What I am referring to here of course is meningo-vascular and meningo-encephalitic syphilis and these infections, specially the meningovascular type of syphilis can sometimes be very acute, with a high fever and extremely severe. There are mycotic organisms like torula which can be picked up if one is very experienced in looking at the spinal fluid and not confusing them with lymphocytes.

In addition there are coccidioidomycosis, and then small parasites. Cysts, I don't think we need mention. As far as virology is concerned I don't know anything about it except I think I would rather leave that for one of the laboratory people to discuss. The mode of spread is perhaps of some interest.

I think the viruses probably go by way of airborne infection or water-borne infection. Rickettsial infections are spread by insects, either ticks or lice or something of that sort. There has been a question in the literature of neuropathology as to where the organism strikes first.

I think the present consensus is that it is carried by way of the small arteries into the brain substance and then leave by way of the perivenous spaces, the Virchow-Robin spaces in the meninges, but that the primary brunt of the infection is born in the brain substance itself.

As I mentioned before, the current treatment of viral encephalitis is unsatisfactory. We have been using chloromycetin and steroids on the notion that they might have something to do with inhibiting edema, correction of respiratory difficulties by means of tracheotomy, combating gastric dilatation with gastric tube and suction and general supportive measures such as transfusion. I stated my idea about the diagnosis before and now we will find out about the real facts.

Dr. McNeil:

It would be difficult to enlarge or increase on that excellent and complete discussion of all the various possibilities in differential diagnosis. If this is the case I am thinking it is, I saw the patient and at the time I must admit it seemed much more simple. The child had an acute, rapidly progressive disease.

There was predominantly coma, no true paralysis, apparently diffuse cerebral disease without any real localization. As it was admitted at a time when Dona Ana County was receiving national recognition for encephalitis, it seemed to us the diagnosis was pretty obvious. I will be interested to see what Dr. Bornstein says.

Dr. Licon:

I would like to ask a question of the eminent persons here present whether this disease is carried by mosquitoes.

Dr. Bornstein:

I think it has been proven that encephalitis, as well as polio, has an insect vector. I remember a polio epidemic in Centralia, Ill. The disease frequency in certain areas had definitely something to do with drainage and the appearance of mosquitoes. Not that this is the exclusive mode of transmission, I am not sure about that, but I am quite sure that there is a mosquito factor.

Dr. Verosky:

The house officers always think I am an old maid about details on history, but I would think that the most common cause of central nervous symptoms in a child with gastroenteritis is dehydration. The protocol doesn't, and I will just bet the chart doesn't say just how much vomiting, how much diarrhea there was.

Diarrhea is so often a constitutional symptom of any sort of infection in infants and children. Unless you dig around in the details of it, it is either a red herring or you are omitting something that could be very important.

Loose Stools

When a mother reports that the child has loose stools, it doesn't mean a thing until you find out how many stools, for how many days, what color, how did they smell and was there any blood.

These details can be extremely helpful in deciding whether or not the diarrhea is a constitutional symptom like fever, which children so often present, or if it is the primary disease. To recognize diarrhea, as a result of disease, gastroenteritis, the biggest help in it is the smell.

They smell terrible. The diarrhea that occurs with acute appendicitis, that occurs with otitis media, with respiratory infections and so forth does not. You don't have to look very hard, you can tell by the look on the mother's face when you ask her, "How does it smell?"

If this were real gastroenteritis, then I think the most likely possibility here is a stroke. Children with dehydration from vomiting and diarrhea of any severity frequently get a cerebral thrombosis. A stroke can certainly give you xanthochromic fluid, it can give you an elevated protein, and it can give you some cells.

It will of course produce a normal sugar. Many of those thromboses are accompanied by secondary hemorrhage but not all. These strokes can kill, these strokes can cripple. Permanent paralysis can result therefrom.

If I were told from the protocol that the patient had a lot of vomiting, a lot of diarrhea, I would certainly place that consideration first.

It takes a fairly severe gastroenteritis to dehydrate an 11 month old baby to the point of danger from central nervous system symptoms or death from shock, but it can happen.

Dr. Stern was talking about the hematocrit. This is normal depending on your standard of reference. In this hospital in a Latin-American child of clinic clientele, that is a pretty good hemoglobin. It is not normal, but when we see that on the ward we do not think of a bleeding disease or something producing anemia, because most of the kids in the clinic walk around with 9.5 to 11 grams hemoglobin

In the long list of viral possibilities we should think of coccackie viruses. They can produce encephalitides and also give myocarditis. If the patient had myocarditis, coccackie virus would certainly be a possibility. One other thing before I leave the point of gastroenteritis, the most common cause of diarrhea which produces convulsions and high fever of course is samonella.

Clinical Diagnosis: Poliomyelitis

Dr. Stern's Diagnosis: Acute encephalitis.

Pathological Diagnosis: Acute hemorrhagic encephalitis.

Pathological Diagnosis by Dr. Bornstein:

On autopsy the organs of the chest and abdomen were essentially negative. There was no enteritis. There were no mural thrombi or other lesions outside the brain which could help in the interpretation of this case. The brain as a whole was large and swollen.

There were numerous hemorrhagic areas which

Figure 1. Gross picture showing extensive hemorrhagic change of brain.

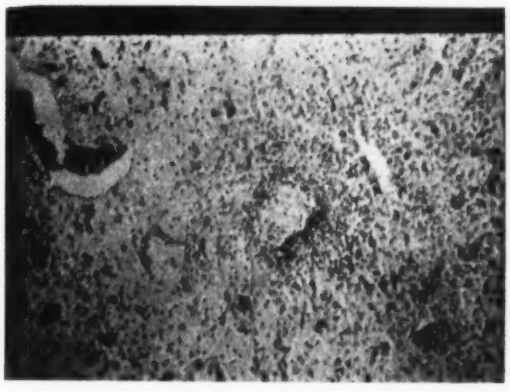

Figure 2. Microscopic picture showing hemorrhagic change of brain associated with diffuse inflammation.

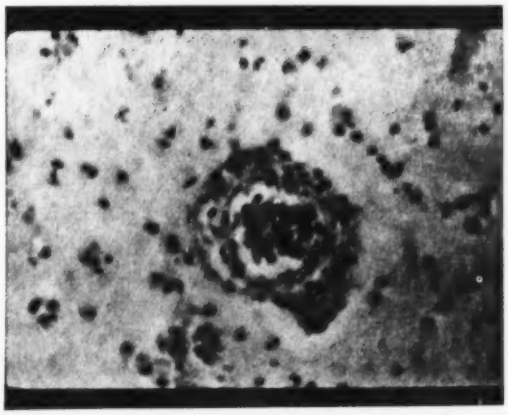

Figure 3. Microscopic picture of brain showing arterial damage.

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measured up to several cm. in greatest diameter, the largest one in the posterior region of the cortex on the right side. Some of these areas were firm, sharply circumscribed and nodular, and suggested the presence of a space-occupying lesion. (Fig. 1). In addition, on sectioning one saw innumerable punctate and confluent hemorrhages throughout the brain substance.

On microscopic examination there were areas of hemorrhage. In addition, there were areas where the hemorrhage was organized around blood vessels and a halo of inflammatory cells was noted around these blood vessels. (Fig. 2).

This was a diffuse lesion, not a polioencephalitis which implies gray matter only, but a lesion which was present in the gray as well as the white substance of the brain.

In some areas where the destruction wasn't too extensive, one could see a typical endarteritis of the smaller branches which were surrounded by a halo of mononuclear leukocytes and plasma cells (Fig. 3).

It is difficult to interpret these findings. The older literature was very free with the term of so-called hemorrhagic encephalitis, using it for lesions which we now consider vascular degenerative in etiology. In the newer articles one does not find too much about hemorrhagic encephalitis except with reference to those lesions which are secondary to infections such as the rickettsial group.

However, with a picture as here, where there is an endarteritis with occlusion of the lumen and inflammatory exudation around the arteries with toxic damage all over, I think one is justified in talking about encephalitis and can accept the large hematomas as secondary to the inflammatory process.

In the absence of any purulent changes and in the absence of any local changes outside the brain, this then can be accepted as a primary, genuine, hemorrhagic encephalitis, probably due to a virus.

Dr. Stern made quite a point out of the fact that this child acted decerebrated. I think the diffuseness of the lesions with damage to the entire brain substance correlates well with this clinical observation. In summary, then, we have here a case of an acute hemorrhagic encephalitis, probably due to a primary virus infection of the brain.

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